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THE BRITISH JOURNAL
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JULY, 1943

COMMUNICATIONS

CHOROIDAL DETACHMENT

BY

B. W. RYCROFT, R.A.M.C.

LATE LANG RESEARCH SCHOLAR, MOORFIELDS

LONDON

INTERNATIONAL ophthalmology has recently given much thought to the treatment of detachment of the retina. This paper deals with a poor relation, namely detachment of the choroid, and like the poor relation it occupies a position of insignificance in text-books where it is as often as not ignored entirely or briefly dismissed with a few words. Yet, like the poor relation, although in the background, it may be the cause of a great deal of trouble. In 1868 Knapp reported the history and pathological examination of a case of choroidal detachment which he had removed by mistake for sarcoma. It occurred in the era when the classical iridectomy was much in favour for acute glaucoma and it is little wonder that many eyes came to be enucleated for tumour when, after the oedema of the cornea had improved following an iridectomy, a choroidal detachment became visible. Several eyes also suffered the same fate in connection with cataract about that time. Graefe and Liebreich were early observers, along with Michel (1878), Beyes (1878), Hjort (1882), Hay (1880), Groenouw, Elschnig (1891), Story (1891), and Devereux Marshall (1895). Their cases were all associated either with cataract extraction or with iridectomy which by this time was in general use. In 1900 the interest of Fuchs resulted in a classical paper and by 1902 he had seen 37 cases of choroidal

detachment which had occurred on 23 occasions after 493 cataract extractions and on eleven occasions after 14 iridectomy operations for acute glaucoma. The trephine at this time was not being used to any great extent. He formulated a theory that the aqueous passed through a rent into the suprachoroidal space separating the choroid and retina together.

This was not entirely accepted, notably by Dor: another school supported the theory of choroidal exudation. Further pathology was discussed by Augstein (1901), Biette (1901), Axenfeld (1902) and Demaria (1904).

The filtration age of glaucoma subsequently followed with the advent of the Elliot trephine and the Lagrange sclerectomy.

Schur in 1913 reported on the occurrence of three cases of choroidal detachment following the Elliot trephine operation. Maschler in 1920 had previously reported choroidal detachment of a persistent type which followed a trephine operation, whilst Hagen in the following year had found that 76 per cent. followed the trephine operation and 22 per cent. were noted in cataract cases in an analysis of 66 cases of choroidal detachment.

In 1930 Rubert systematically examined a series of 50 cases of chronic glaucoma 48 hours after trephine operation at Riga and found that 50 per cent. showed detachment of the choroid: he remarked that the separation was peripheral and more often nasal than temporal and more pronounced in elderly people.

By this time, an opinion was rapidly gaining support that choroidal detachment was an occasional occurrence with an invariable good prognosis. Up to 1921 experimental work was limited to that of Meesman who was able to demonstrate choroidal detachment in the absence of high tension, by means of a persistent corneal fistula, resulting in hypotony and choroidal exudation.

More recently in 1934, Dor described a case of glaucoma in which a bilateral trephining was carried out. A choroidal detachment occurred two months after the operation in one eye and against his own convictions but in deference to those of several colleagues, he enucleated the eye for sarcoma thereby repeating the mistake of his father in 1896. His conclusions were that choroidal detachment is of much greater frequency than is generally recognised; that it is often confused with choroidal sarcoma, and that diascleral transillumination is no certain guide in differential diagnosis. Pathological appearances showed interstitial choroidal haemorrhages, general oedema of the choroid, and sub-choroidal exudation without blood. Other spontaneous choroidal detachments have been described by Meller, Fleischer, Bussy and Würdemann.

And so to the present time, when sterility in the literature is largely responsible for the discrepancies which occur in international text-books, some giving a good prognosis, others a bad prognosis, whilst in others no mention at all is made.

There are several theories of which mention only is made as to the cause of these choroidal detachments. There is that of Haab who believed detachment to be a cyst; of Velhagen that there were cyst-like detachments in the epithelium of the ciliary body and of Lindemann that the detachment was a swollen and blood-stained cortical mass in the vitreous. Hudson's theory is that a serous exudate arises from the choroidal blood vessels, particularly from the choroidal veins and that under conditions of abnormal low tension, aided by negative pressure, free fluid transudes which is absorbed when the tension becomes re-established. These views are not entirely acceptable to-day. The theory of Fuchs seems to be the most rational, but one objection to this is that Heine has observed only one case of choroidal detachment in many hundreds of cases of cyclodialysis.

Barkan lists four classes of choroidal detachment:

1. Tear of the ciliary body with the aqueous percolating into the suprachoroidal space. (Simple type).

2. Traction on the choroid and retina by organising masses in the vitreous. (Traction type).

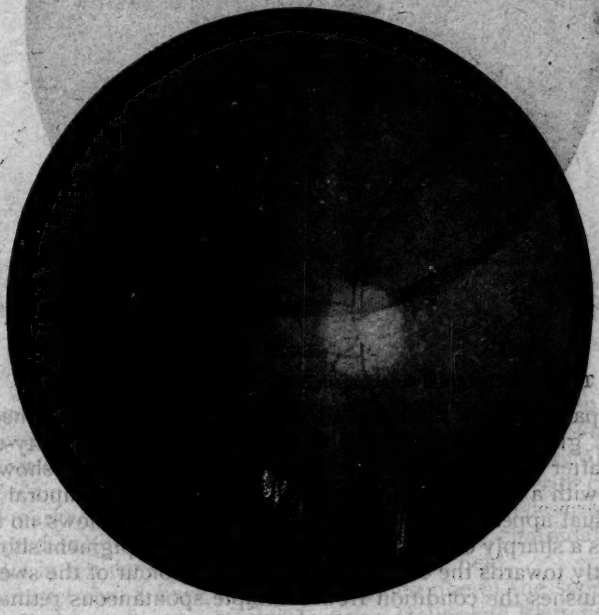

FIG. 1.

Simple choroidal detachment 48 hours after trephine operation.

3. Sub-choroidal haemorrhage. (Degenerative type).

4. Massive choroiditis with sub-choroidal exudate. (Inflammatory type).

The choroidal detachment which is the subject of this paper in particular is the first or simple type.

Meller has classified this simple type of choroidal detachment into (i) immediate, (ii) delayed, and (iii) spontaneous.

The following case is an example of the immediate and common type. It is illustrated by Fig. 1.

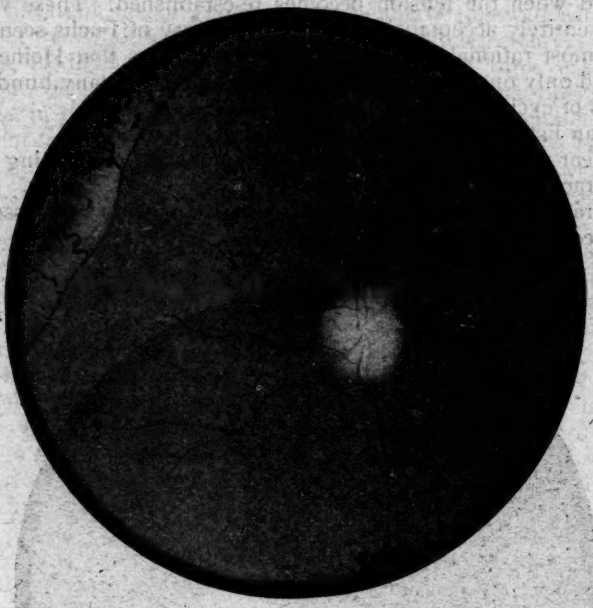

FIG. 2.

The same choroidal detachment 3 days after trephine operation.

The patient was a woman of 61 years who had been trephined for chronic glaucoma simplex by the classical method. Forty-eight hours after operation the condition of her eye was as shown in Fig. 1 with a large detachment of the choroid in the temporal half. The usual appearance is well seen. The swelling shows no folds and has a sharply cut edge; occasional patches of pigment show up distinctly towards the ora serrata. The dark colour of the swelling distinguishes the condition from a simple spontaneous retinal detachment. No holes or tears are seen. At this time there was no filtration bleb and no formation of the anterior chamber. Fig 2

shows the condition three days later and by the end of the week the detachment had disappeared, coincident with re-formation of the anterior chamber and good filtration.

The second type of Meller's classification is the delayed choroidal detachment which is illustrated by Fig. 3. This patient was a man aged 64 years who had been operated on by double trephine for glaucoma three years previously. The result had been satisfactory but suddenly there came a deterioration of

FIG. 3.

Delayed choroidal detachment 3 years after successful trephine operation.

vision probably caused by the rupture of an ectatic bleb. Fig 3 shows the condition which was found at that time. There was almost a total peripheral choroidal detachment except for an area at "12 o'clock." With this there was hypotony, no anterior chamber, and marked miosis. Operative treatment, as will be described later, resulted in the complete disappearance of these choroidal detachments in ten days with the restoration of the field.

I regret that I have no example to describe of the spontaneous type as these cases are extremely rare. Meller and Fleischer report the only cases which I have been able to find.

It was decided to carry out experimental and clinical investigations into the cause and treatment of choroidal detachments in animals and man. In animals, an endeavour was made to produce detachment of the choroid by trephining a cat's eye at various levels of increased intra-ocular pressure.

The cat was anaesthetised by chloralose and the tension of an eye raised to 40, 60 and 80 mm. Hg by injecting saline into the vitreous chamber.

A 2 mm. trephine opening at the limbus was made in the usual manner and the choroid examined ophthalmoscopically at half-hour intervals for four hours, and subsequently on the following three days. Results were unsatisfactory and no detachment of the choroid was produced in any animal.

In man the routine procedure was to examine the fundus of every case of trephine, cataract, and iridectomy within 48 hours of operation. In all there were sixty eyes with cataract extraction, both of extra and intra-capsular type, and eighty eyes which had had a trephine operation. Six were cases of iridectomy for sub-acute glaucoma. Two choroidal detachments were noticed amongst the

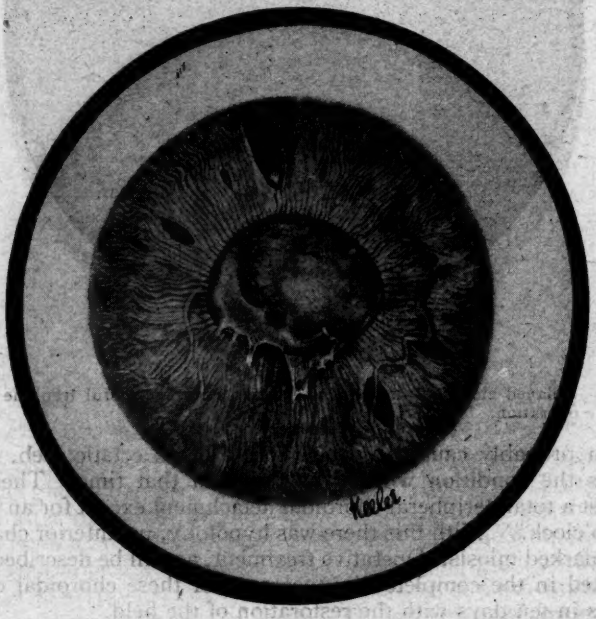

FIG. 4.

Result of prolonged detachment of the choroid and delayed reformation of anterior chamber.

cataract cases in the extra-capsular group, both of the immediate type and in the upper temporal area, and both subsided without untoward result. In the eighty trephine cases, 18 showed choroidal detachments of various sizes three days after operation. Of these 18 cases two resulted in a long convalescence with delayed reformation of the anterior chamber and hypotony. One healed without filtration and the other showed capsular changes and subsequent lens opacity. Fig. 4 illustrates the unfortunate end-result of the latter case. The tag from the anterior capsule passing to the posterior surface of the cornea is seen below. In this case the anterior chamber did not reform for three weeks and persistent hypotony was maintained. In one case of absolute glaucoma an experimental trephine was done and a choroidal detachment was produced by sub-choroidal haemorrhage, resulting in hetero-chromia of the iris from blood staining and persistent subnormal tension. No choroidal detachment was noted after broad iridectomy in the four cases of acute glaucoma.

Gradle has reported two cases lasting three weeks following detachment of the retina treated by Larssen diathermy. I have not been able to discriminate this type of choroidal detachment.

As the result of investigations into this condition the following conclusions as to aetiology have been reached :—

1. That detachment of the choroid is primarily due to a forward movement of the diaphragm, consisting of ciliary body, suspensory ligament and lens. This probably results in a tear near the ora serrata. In order that such a movement forward be possible, there must be a sudden loss of the anterior chamber and to my mind this is the primary cause and not the result of a choroidal detachment. In the case of a trephine operation, a choroidal detachment of the immediate type is always associated with a leak either from the conjunctival flap or from the trephine bleb. It was possible to demonstrate this frequently by Seidel's fluorescein test. The fluorescein, whilst flowing over the conjunctiva remained green, but as soon as it came in contact with the leaking aqueous from the flap, precipitation occurred and a fine streak of the usual bright green colour could quite easily be detected.

2. Individual technique in the trephine operation has no bearing. It occurs in cases of 2mm. and 1½ mm. trephines, with complete iridectomy or with peripheral iridectomy, and irrespective of initial tension. My feeling is that the reason a choroidal detachment occurs in some trephines and not in others is due to a difference in the depth of the anterior chamber. It is more prone to occur in those cases of chronic glaucoma where the anterior chamber is deep, thereby allowing a greater forward movement of the iris-lens diaphragm.

3. When the peripheral tear in the choroid heals, the aqueous

then passes into the anterior chamber. If the conjunctival flap is sealed and has no leak, the anterior chamber re-forms and the filtration bleb appears. The fluid in the choroidal detachment is subsequently absorbed.

Treatment of choroidal detachment is almost generally ignored in all text-books. In a personal communication with the late Colonel Elliot, he said that he felt the best thing to do was to get the patient out of bed, and was of opinion that most cases righted themselves. Meller reports one case treated by diathermy, while Verhoeff has suggested scleral puncture to release the sub-choroidal fluid. This has not been found to be successful and that is to be expected.

The most satisfactory form of treatment seems to be associated with a closure of the fistula in the conjunctival flap. In the case of delayed choroidal detachment (Fig. 3) all that was done was to demonstrate a leak close to the trephine hole. This was subsequently covered by a vizor conjunctival flap and the choroidal detachments slowly absorbed within ten days.

There are one or two sequelae of choroidal detachment which must be borne in mind. First of these is the fact that when a trephine case has had a choroidal detachment of prolonged duration the trephine bleb is usually flat and filtration is subsequently scanty. Secondly if the anterior chamber remains unformed for longer than a week, an attempt should be made to seal the leak and to refill the anterior chamber with saline, as there is then a real risk of anterior capsular changes by contact.

These investigations were carried out in 1938 during my tenure of the Lang Research Scholarship at the Royal London Ophthalmic Hospital, to the Trustees of which I express my gratitude. Delay in publication is due to war service.

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THE RELATION OF RIBOFLAVIN TO THE EYE. A REVIEW ARTICLE

BY

A. PIRIE

NUFFIELD LABORATORY, THE EYE HOSPITAL, OXFORD

RIBOFLAVIN became of immediate interest to ophthalmologists after the publication by Sydenstricker, Sebrell, Kleckley and Kruse (1940) of the paper in which they describe the ocular signs of ariboflavinosis in man. In fact, so much interest was aroused that a year later the Science News Supplement to Science (1941) carried a report from an eye specialist in Maine that 80 per cent. of people examined showed evidence of both past, chronic or new signs of deficiency of riboflavin. If the eye specialist made a return trip at the present time it is doubtful if he would reach the same conclusion, since the last two years have been spent in defining the true signs of riboflavin deficiency in man and animals.

Uncomplicated riboflavin deficiency in man probably never occurs. The diet is, in practice, always deficient in more than one of the vitamin B complex constituents. In the series of cases described by Sydenstricker, nicotinic acid, thiamin, ascorbic acid and cod liver oil were added to the diet in order to make the deficiency of riboflavin the dominating one. Ocular signs may be noticeable before other disorders. The patient seems usually to complain of photophobia and dimness of vision, and on examination circumcorneal injection is seen, and, to quote Sydenstricker *et al.*, "The earliest change that can be recognised with the slit-lamp is marked proliferation and engorgement of the limbic plexus with the production of great numbers of very narrow capillary loops which outline the extreme margins of the scleral digitations and obliterate the normal narrow avascular zone between the plexus and the sclerocorneal junction. . . . The cornea is actually invaded first by very small capillaries arising from the apexes of loops surrounding the scleral digitations. . . . Such capillaries lie just

beneath the epithelium and soon anastomose to form a tier of loops from which more single capillaries arise, extending centripetally." At a later stage the deeper layers of the cornea may be invaded by vessels and superficial and interstitial opacities may develop. After giving riboflavin, 5-10 mg./day, the photophobia may clear up within 24-48 hours and in this time also the engorgement of the limbal vessels subsides. If vessels have penetrated any distance on to the cornea 8-15 days treatment is necessary for their emptying. In all instances the vessels remained visible as greyish or refractile streaks.

Sebrell and Butler (1939) described in detail the general signs of ariboflavinosis in man. The lips become abnormally red and cracks may appear at the angles. There is seborrhoea of the nasolabial folds, eyelids and ears, and there may be seborrhoea and follicular keratosis of the forehead, malar eminences and chin. One of the very early signs is glossitis. The tongue is clean, purplish red and sometimes fissured, with large and flattened or mushroom shaped papillae. Sydenstricker *et al.* report that they have seen this type of tongue appear in pellagrins who had been cured of their pellagra with nicotinic acid, but remained deficient in riboflavin.

It is the attributes of superficiality and of symmetrical arrangement which mark off corneal vascularisation due to deficiency of riboflavin from the corneal vascularisation due to other vitamin deficiencies and pathological states. Johnson and Eckhardt (1940) reported that riboflavin improved rosacea keratitis, where the corneal vessels are irregular in distribution and in depth. Recently Fish (1943) has reported that in her hands riboflavin has no beneficial effect on rosacea keratitis. Kruse *et al.* (1940) found an improvement in four cases of syphilitic keratitis after riboflavin. Here again the vessels are of a different type from those seen in the ariboflavinosis of man and animals. Wagener (1941) commented on this and stated that he was unable to obtain any improvement of syphilitic keratitis with riboflavin therapy. This fundamental question of the diagnosis of riboflavin deficiency by an appraisal of the type of vascularisation seen is fully dealt with in a paper by Gregory (1943). It is not the purpose of this review to go deeply into the picture of riboflavin deficiency in man, but the papers already referred to show that there has been considerable confusion and it is possibly suitable to make a short survey of all the experimental data connecting riboflavin with the eye and to outline in brief the rôle it is thought to play in tissue metabolism.

Chemistry and Physiology of Riboflavin.—Riboflavin has now been firmly established as vitamin B_2 and has been found to be an essential part of the oxidising systems of animal tissues. It has been synthesised and its structure has been established as 6·7.

dimethyl 9. d-i ribityl iso-alloxazin. The work, which led up to the establishment of these facts has already been described in many reviews and the description will not be repeated here. References to such reviews are given at the end (Fig. 1).

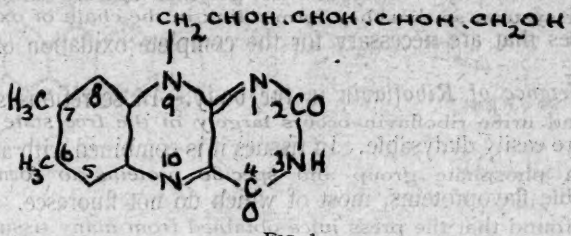

FIG. 1.

Riboflavin.

The recent advances in our knowledge of the relation between riboflavin and tissue oxidation must be described. The account will not be presented in strict historical sequence, but rather the present position will be described in terms of the earlier work.

Properties of Riboflavin.—Riboflavin is a bright yellow solid. It is slightly soluble in water, giving bright yellow solutions which show a very strong green fluorescence. The concentration of riboflavin in a solution can be estimated by determining the strength of this fluorescence. In solution riboflavin is heat stable, but it is fairly rapidly destroyed by light. The destruction is most rapid in ultra-violet light, but daylight is also slowly destructive, so that solutions of riboflavin must be preserved in the dark. In neutral solution the effect of light is to split the ribose side chain off the molecule. The resulting substance, lumichrom, still fluoresces weakly green-yellow, but has no vitamin activity and cannot replace riboflavin in the diet. The effect of light on alkaline solutions of riboflavin is different. Under these circumstances the four terminal carbon atoms of the ribose side chain are split off, giving a substance with a bright blue fluorescence, called lumiflavin, which again has no vitamin activity. The structure of riboflavin was partly elucidated from a study of these photolytically produced breakdown products of the vitamin.

Solutions of riboflavin can easily be reduced by sodium hydrosulphite or palladium and hydrogen. The reduced solution is no longer bright yellow in colour, but the yellow colour can be restored and the riboflavin re-oxidised by shaking the solution with air or oxygen. This process of reduction and re-oxidation can be carried out indefinitely. Reduced riboflavin has combined with 2 H atoms which are linked to the N atoms at positions 1 and 10

of the iso-alloxazin rings and when the solution is shaken with air these hydrogens are given up to oxygen. This reversible reduction of riboflavin serves as a model for its most important action in the body. In the tissues riboflavin compounds are constantly being reduced by one system of enzymes and re-oxidised by another system, so that they form a link in the chain of oxidation processes that are necessary for the complete oxidation of metabolites.

Occurrence of Riboflavin in the body.—In secretions such as milk and urine riboflavin occurs largely in the free state and is therefore easily dialysable. In tissues it is combined with adenylic acid, a phosphate group and specific proteins to form non-dialysable flavoproteins, most of which do not fluoresce. Pulver (1940) found that the press juice obtained from many tissues contained very little of the tissue riboflavin. Prolonged dialysis may release riboflavin from tissues but this is probably due to the breakdown of flavoproteins. Estimation of riboflavin in tissues depends either on its property of fluorescence, a typical method being described by Najjar (1941), or on the fact that the growth of certain bacteria is proportional to the concentration of riboflavin in their medium. This microbiological method worked out by Snell and Strong (1939) is the most satisfactory one at present available and enables 0·05γ riboflavin to be estimated accurately. In general, the liver, kidney and heart of most animal species are the tissues richest in riboflavin, but all tissues so far examined contain small amounts. It is also present in eggs, yeasts and bacteria, and possibly the virus of vaccina. It has been estimated that an adult man needs 3-5 mg. riboflavin per day.

Flavoprotein Enzymes.—Warburg (1938) isolated the first riboflavin containing enzyme from animal tissues. He had earlier (1932) isolated an oxidation enzyme containing riboflavin from yeast, which he had shown to be similar to a yellow enzyme preparation from muscle which Banga and Sz. Gyorgi had made in 1932. Warburg found that preparations of the *d*-amino oxidase from kidney contained a riboflavin compound and finally showed that the enzyme itself is a flavoprotein which contains flavin adenin dinucleotide as its active group. This flavin adenin dinucleotide (Fig. 2) can be separated from the specific enzyme protein. Separately, neither the protein nor the flavin is catalytically active, but they can be recombined by simply mixing the two solutions to give an active enzyme. This enzyme in the presence of *d*-amino acids—that is, the isomer which does not occur in proteins—will take up oxygen, being reduced by the amino acid and constantly re-oxidised by the oxygen of the air.

The fact that this enzyme can be, as it were, taken to pieces and then put together again has proved very useful in investigating

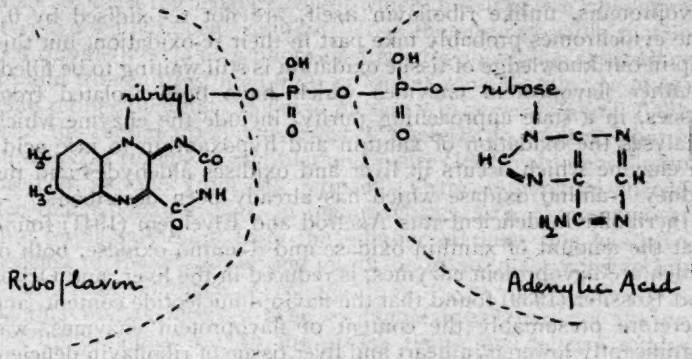

FIG. 2.

Flavin adenin dinucleotide

flavins in tissue extracts. Flavoproteins from other sources were dissociated into flavin plus protein and the effect of this flavin on the oxidation of *d*-amino acids in the presence of the specific kidney protein was then investigated. If the " foreign " flavin compound can replace the kidney flavin and combine with the kidney protein to give an active enzyme it is assumed that this " foreign " flavin is identical with the kidney flavin, namely flavin adenin dinucleotide. In this way flavin adenin dinucleotide has been found to be the active group of all well characterised tissue flavoproteins.

The most important function of the flavoprotein enzymes so far described is the oxidation of the nicotinic acid co-enzymes. Studies of the mechanism of tissue oxidation have shown that many substances are oxidised by specific enzymes, plus co-enzymes which are substances of relatively low molecular weight. Two of the most important co-enzymes contain nicotinic acid amide as the active group. This becomes reduced by the substance undergoing oxidation, but the mechanism whereby it is re-oxidised so that it can again take part in oxidation processes was unknown until Straub (1939) isolated a flavoprotein from pig heart which rapidly re-oxidised the nicotinic acid co-enzymes, becoming itself reduced in the process. We may picture the oxidation of a metabolite as taking place in the following way :—

1. Substrate + enzyme + co-enzyme → oxidised substrate + enzyme + reduced co-enzyme.
2. Reduced co-enzyme + heart flavoprotein → reduced flavoprotein + co-enzyme.
3. Reduced flavoprotein + (?) → flavoprotein + reduced (?).

The way in which reduced flavoproteins are re-oxidised is still unknown; under normal tissue conditions of O_2 tension, most

flavoproteins, unlike riboflavin itself, are not re-oxidised by 0_2. The cytochromes probably take part in their re-oxidation, but this gap in our knowledge of tissue oxidation is still waiting to be filled.

Other flavoprotein enzymes which have been isolated from tissues, in a state approaching purity, include the enzyme which catalyses the oxidation of xanthin and hypoxanthin to uric acid; an enzyme which occurs in liver and oxidises aldehydes and the kidney -amino oxidase which has already been described.

In riboflavin deficient rats Axelrod and Elvehjem (1941) found that the amount of xanthin oxidase and d-amino oxidase, both of which are flavoprotein enzymes, is reduced in the liver, and Ochoa and Rossiter (1939) found that the flavin dinucleotide content, and therefore presumably the content of flavoprotein enzymes, was significantly lowered in heart and liver tissue of riboflavin deficient rats, though not much affected in kidney and brain.

Synthesis of flavin adenin dinucleotide from riboflavin occurs very rapidly after the injection of riboflavin into the animal. Ochoa and Rossiter (1939) found that the flavin adenin dinucleotide content of the liver of riboflavin deficient rats increased half an hour after an injection of riboflavin. *In vitro* they found that liver slices did not synthesise the dinucleotide from added riboflavin but decomposed the dinucleotide already present in the tissue. Klein and Kohn (1940) found that human blood cells carried out the synthesis very slowly, but Trufanov (1941) found that a good many tissues, muscle in particular, could synthesise the dinucleotide. He found that there was a balance between synthesis and breakdown and the synthesis could only be shown if the tissue slices were incubated with riboflavin for very short periods. In longer periods breakdown was the dominating process.

RIBOFLAVIN AND THE EYE.

Occurrence.—Euler and Adler (1934) estimated riboflavin in the eyes of many species of fish and some mammals by a fluorimetric method. They found that the retinae of all species contained riboflavin but they could find none in the other parts of the eye. In some fish the retinae contained very large amounts of riboflavin, larger amounts than had previously been found in any mammalian tissue. Euler and Adler considered that the retinal riboflavin played some part in a light sensitive reaction owing to the fluorescence of free riboflavin. In mammalian tissues riboflavin occurs combined with adenylic acid and proteins and in general these flavoproteins do not fluoresce. In order to utilise riboflavin fluorescence the riboflavin must occur uncombined. Adler and Euler (1938) obtained evidence that free riboflavin does occur in cod retina. Frozen cod retina was extracted with ice water and the

extract shaken with benzyl alcohol, which dissolves free riboflavin. This gave a fluorescent solution. It is therefore possible that in fishes riboflavin acts as a photosensitiser by absorbing short-wave light and transmitting it as light of longer wave length. In mammalian retinae the total riboflavin present is in general about 1/100th that of the retinal riboflavin in fish. There is no evidence that it occurs as free riboflavin as Euler and Adler (1934) could not obtain fluorescent extracts from calf retinae. There is therefore no evidence that riboflavin does, in mammals, act as a photosensitiser in the retina, although from the number of times this has been suggested in the literature one might be excused for considering it a well established fact.

Riboflavin has recently been estimated in all parts of the ox eye, Philpot and Pirie (in the press), using the delicate microbiological method of Snell and Strong (1939). A parallel series of estimations of the flavin adenin dinucleotide content of the eye tissues was also run in order to see whether riboflavin occurred free or combined with adenylic acid. Only traces of riboflavin or flavin adenin dinucleotide were found in the vitreous or aqueous humours, in the lens and in the substantia propria of the cornea. The corneal epithelium, the ocular conjunctiva, the iris, the ciliary body and the choroid (plus pigment epithelium) all contained 2 - 3γ of total riboflavin per gm. wet weight of tissue. Probably all this riboflavin was combined with adenylic acid as flavin adenin dinucleotide. The retina contained 3 - 4γ total riboflavin per gm. wet weight. A large part of this occurred as the dinucleotide, but the agreement between the two different estimations was not as satisfactory as with the other tissues and there is a possibility that a very small amount of riboflavin may occur free in ox retina.

Meibomian glands, lacrymal glands and Meibomian secretion contained more riboflavin than any part of the eye, 4 - 6γ per gm. wet weight. Here again the riboflavin was combined with adenylic acid to give flavin adenin dinucleotide.

GENERAL EFFECTS OF DEFICIENCY OF RIBOFLAVIN IN ANIMALS.

As the complexity of the B vitamins has become greater and greater, so the signs of the deficiency of any one of the constituents of the complex have been narrowed down. But the present position is that members of the complex are thought to interact with one another, so that although one set of signs may be broadly diagnostic of—say riboflavin—deficiency, yet some of these signs may occur in animals receiving adequate riboflavin but deficient in pyridoxin, pantothenic acid or yet unknown factors. This overlap is described by Chick, Macrae and Worden (1940) who kept rats for long periods deficient in one or other of these members

of the B_2 complex. Simple riboflavin deficiency led to a failure in growth and the rats developed an eczematous condition of the skin affecting especially the nostrils and eyes. The hair round the eyes fell out and there was conjunctivitis, blepharitis and dullness of the cornea, the eyelids being stuck together. The fur also became scanty over the head and forelegs and appeared moist and matted with bald patches developing later. In a group of ten rats which received adequate riboflavin, but were deficient in other members of the B complex, seven animals showed—as well as the dermatitis characteristic of pantothenic acid and pyridoxin deficiency—riboflavin deficiency type of skin lesions with conjunctivitis and exudate from eyes and nostrils.

In dogs the general signs of riboflavin deficiency are lack of appetite, weakness and ataxia with sudden collapse and death after about four months on the diet. If riboflavin is given in the terminal stages it produces a rapid cure. In chicks, prolonged partial deprivation of riboflavin has been studied and it has been found to lead to " curled-toe paralysis " due to degeneration of the sciatic nerves. Acute riboflavin deficiency in the chick leads to paralysis and death as in the dog. The same picture has been described for the pig.

Effect of deprivation of Riboflavin on the Eyes of experimental Animals.—That changes occurred in the eyes of rats kept on a riboflavin deficient diet was first noticed by Day, Langston and O'Brien (1931). These workers noticed that there was vascularisation of the cornea and also found that nearly all their animals developed cataract. They considered that the cataract was due to the deprivation of riboflavin and in the next few years published a series of papers describing such cataracts in rats, mice, chickens and monkeys, and showing that they could be prevented or arrested by the addition of riboflavin to the diet, although a cataract could not be cleared up by riboflavin. These results caused great interest and workers in other laboratories started work on the same lines. In general, no one obtained 100 per cent. cataract as Day *et al.* had observed in their animals. Bourne and Pyke (1935) got 20-30 per cent. cataracts. Eckhardt and Johnson (1939) obtained an even smaller percentage and Gyorgy (1935) and Bessey and Wolbach (1939) found no cataracts in their experimental animals. Naturally each group of workers has used a slightly different diet and all the rat strains have not been the same. It is therefore possible that the divergent results are due to slight divergences of technique. At present it seems unlikely that such cataracts are caused by a simple deficiency of riboflavin. Recently Baum, Michaelree and Brown (1942) have claimed that rats on a diet absolutely free from riboflavin fail to develop cataract, whereas 70 per cent. of rats on the same diet plus very small amounts of riboflavin develop

cataracts. Stocksted and Manning (1938) found that chicks on a completely riboflavin free diet did not get " curled-toe paralysis " so frequently as chicks which received minimal amounts of riboflavin. They concluded that this was due to the stimulation of growth by these small amounts of riboflavin, the need for riboflavin and all other members of the B_2 complex becoming thereby much greater. It is possible that formation of cataracts in riboflavin deficient rats depends on imbalance between the different members of the B_2 complex.

Corneal vascularisation, first noticed by Day, Langston and O'Brien, has been found by Bessey and Wolbach (1939) to be one of the earliest and most constant signs of riboflavin deficiency in rats. They have described its development in detail and the parallelism between their description of the vascularisation of rat cornea and the vascularisation of human cornea in a-riboflavinosis described by Sydenstricker *et al.* (1940) is very striking indeed. The eye changes have not been described in such detail in other experimental animals, but Patek, Post and Victor (1941) described a clouding of the pig's cornea after five months deficiency of riboflavin. No slit-lamp observations were made, but histological preparations showed that the corneal epithelium was cornified on the surface with the lower cells swollen and irregular. The corneal stroma was more normal.

The cornea is not the only part of the eye affected by riboflavin deficiency. Nearly all observers have noted that, in rats and pigs at least, changes occur in the conjunctiva and lacrymal glands also. Sections of rats' lacrymal (?Harderian) glands were reported by Sherman and Sandel (1931) to show an " acute inflammatory process." Diminished lacrimation has been noted in rats by Shaw and Phillips (1941) and increased lacrimation in dogs by Street, Cowgill and Zimmerman (1941). In pigs the eyelids are swollen and the palpebral fissures narrowed. In severe deficiency in the rat there is conjunctivitis and blepharitis and the lids may stick together completely. Since the lacrymal and Meibomian glands and the Meibomian secretion, at least in the ox, contain a greater percentage of riboflavin compounds than other parts of the eye, it is reasonable to suppose that a deficiency should lead to pathological changes in them. The time relations between these changes and the vascularisation of the cornea has not been described.

It is interesting to notice the long time-lag between the first reports of eye changes in rats on riboflavin deficient diets, the application of this knowledge to human problems and the accurate detailed description of the lesion in experimental animals. Vascularisation of the rat cornea was first noticed in 1931, but was not described in detail until 1939. In 1940 an identical picture was

described in man. In 1939 descriptions of riboflavin deficiency in pigs and dogs make no mention of eye lesions, but in 1941 a description of the lesions in these species is given. No account has yet been given of eye lesions, if any, in riboflavin deficient chicks. There has been in the past a regrettable tendency on the part of experimentalists to neglect the detailed examination of eyes and of ophthalmologists to neglect the pointers given to them by experimentalists.

DISCUSSION.

We do not know how the riboflavin content of blood and tissues changes in riboflavin deficiency, so that it is impossible to speculate whether the ingrowth of vessels into the cornea is called forth by deficiency of riboflavin in the cornea itself or by deficiency of riboflavin in the blood. It has been assumed that the riboflavin of the cornea is derived from the blood, but it is possible that it is partly derived from the eye secretions which do, in the ox, contain flavin adenin dinucleotide. In other tissues riboflavin has been shown to take part in normal oxidation processes and we may assume that it fulfills the same function in the tissues of the eye, but no data of the flavoprotein enzymes of the eye are available. As it has a specific function in the cell it can only be of use when that specific function is upset and it is therefore useless to hope that riboflavin will help conditions where conjunctival hyperaemia or corneal vascularisation are due to other causes.

It is extremely interesting that the eye signs in riboflavin deficiency are some of the earliest to devolop and may be used—if possible in conjunction with skin and tongue changes—to make an early diagnosis of the deficiency. It is the early stages of deficiency which are most likely to be met with in this country, but in tropical countries reports of severe eye lesions associated with multiple vitamin deficiencies have been frequently reported. Pock Steen (1939) reported that sprue patients had lesions in their eyes which caused "twilight blindness" and that this condition was relieved by riboflavin. Landor and Pallister (1935) found that prisoners in Johore suffered from multiple deficiences and among their symptoms were photophobia, dimness of vision, "conjunctivitis and corneal ulceration," cured by Marmite or liver. Similar pictures have been reported by Moore (1939) from Nigeria, where school children were very largely affected, and Metivier (1941) from Trinidad and there is no doubt that eye lesions due to multiple vitamin B deficiencies are very widespread. The clear picture of riboflavin deficiency has been established by Sydenstricker and subsequent workers, but it yet remains to be seen what rôle, if any, is played by each of the other known and unknown members of the vitamin B complex.

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A NOTE ON THE SPHERICAL EQUIVALENT OF SPHERO-CYLINDRICAL LENSES

BY

W. J. B. RIDDELL

GLASGOW

AT a time when it is necessary to exercise simplicity in the prescribing of glasses it may be of value to draw attention to a simple application of the principle of Sturms' conoid. There is a rule of sphero-cylindrical equivalents which was described by Copeland and referred to at some length by Prangen (1937) in a valuable review. The latter defines the spherical equivalent of any formula for the correction of astigmatism as being equal to the value of the spherical lens plus half the value of the cylinder added algebraically. If the cylinder is reduced by any given amount, half of this should be added to the existing sphere. This enables a correction to be chosen which corrects the interval partially. I have used this method for a number of years and have derived great assistance from it, particularly in the refraction of school children and aphakic patients.

TABLE I

Sphere	Cylinder	Spherical Equivalent
+12	—	+12
+11·5	+1	+12
+11	+2	+12
+10·5	+3	+12
+10	+4	+12

An example of the distribution applicable to an aphakic cataract patient is shown in Table I. It is seen immediately that modification of the cylindrical correction must frequently be carried out in practice without the prescriber being aware of what is being done.

The procedure has a useful practical application in patients with mixed astigmatism in whom it may be desirable to under-correct the astigmatic portion of the error. The final decision is one resulting from the combination of many personal factors and, although experience may be the best rule, guidance can be obtained from consideration of the spherical equivalent. An example is shown in Table II giving possible corrections for a patient with

TABLE II

Sphere	Cylinder	Axis	Spherical Equivalent
−2·0	+5·0	90°	+0·5
−1·5	+4·0	90°	+0·5
−1·0	+3·0	90°	+0·5
−0·5	+2·0	90°	+0·5
—	+1·0	90°	+0·5
+0·5	—	—	+0·5
+1·0	−1·0	180°	+0·5
+1·5	−2·0	180°	+0·5
+2·0	−3·0	180°	+0·5
+2·5	−4·0	180°	+0·5
+3.0	−5·0	180°	+0·5

a refraction of +3·0 D. horizontal and −2·0 D. vertical. As in the previous example it will be seen that the method may have been carried out without the observer being conscious of the procedure.

As a result of the war, simplification of civilian prescriptions has been requested by the Council of British Ophthalmologists and the prescribing of eighth dioptre powers is deprecated. It will frequently be found in practice that an equivalent lens calculated upon the basis described will prove to be perfectly satisfactory. Even in peace-time patients sensitive to such refined

corrections were uncommon. An example showing the result of eliminating eighth powers is given in Table III.

TABLE III

Sphere	Cylinder	Axis	Spherical Equivalent
+0·37	−0·75	180°	0
+0·25	−0·5	180°	0
+0·12	−0·25	180°	0
0	0	0	0
−0·12	+0·25	90°	0
−0·25	+0·5	90°	0
−0·37	+0·75	90°	0

It is quite a simple matter to construct a graph designed to satisfy the equation

$$x = a + \tfrac{1}{2}b$$

where x = the spherical equivalent, a = the spherical power and b = the cylindrical power of a sphero-cylindrical lens. If the scales are drawn on squared paper, a ruler laid across the appropriate values will enable the equivalents to be read off rapidly.

On showing the draft copy of this note to a colleague, he suggested that an appropriate sub-title would be "Is your cylinder really necessary?" Without subscribing to this extreme view, there is little doubt that, if attention is paid to the method advocated, the final prescription may be simplified and comfort given to many astigmatic patients.

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TRAUMATIC OEDEMA OF THE MACULA

BY

MAJOR SEYMOUR PHILPS, R.A.M.C.

LONDON

TRAUMATIC oedema of the macula, or commotio retinae, was first described by Noyes in 1871. Many instances have been reported since then, especially following the last war, Salzmann (1919) finding eight cases in 2,400 war injuries, and Middleton (1919) 23 in 100,000. No apology is offered before presenting the following four cases, for it is a condition that is not uncommonly missed because it cannot be seen until the pupil has been dilated.

The condition arises following a blow on the anterior pole of the eye, the macula being the point of contre-coup. More rarely it has apparently followed a blow on the side of the eye. The patient will complain of misty vision and examination of the macula will reveal the typical grey coloration with a central orange-red pit at the site of the fovea. The swelling may subside in a few days in which case some central vision may return, or it may persist for some weeks and in such a patient the outlook for useful vision is very bad, and marked pigmentary changes will be found at the macula. In some patients the oedema does not subside, and after many months or even years the atrophic retina breaks away at the edge of the pit leaving a hole at the macula which has the appearance of having been punched out with a trephine. If this should happen, a retinal detachment is likely to be the end result. The following recent cases are recorded not because they throw any new light on the subject, but because they demonstrate these four stages in the progress of macular oedema: 1. Oedema of the macula. 2. Oedema replaced by pigmentary changes. 3. Hole formation. 4. Retinal detachment.

CASE 1. E.L., aged 35 years, Belgian refugee, July 27, 1942.—While working on demolitions, a wall collapsed on him. Admitted to hospital semi-conscious and with a large haematoma of the right orbital region. X-rays showed a fracture of the vault of the skull, R. side, extending into the anterior fossa. The right optic disc was swollen and eventually became atrophic with loss of all vision in that eye. When this patient recovered he found that he could not see well with the left eye and examination of it revealed the typical appearance of macular oedema (Fig. 1). The vision of this eye was 6/60, not improved by lenses, and neither the fundus appearance or the vision had improved when he was last seen.

CASE 2. Sergeant H.L.G., aged 28 years.—Had always had good vision until twelve years previously, when he was struck a violent blow in the left eye. After that he could never see well with that eye, and reading vision never returned. On examination the vision in this eye was less than 6/60, and the macula showed pigmentary changes such as one would expect to find after the subsidence of traumatic oedema (Fig. 2).

CASE 3. J.S., aged 29 years, a soldier.—Four years previously he had had a severe motor-bike accident and was unconscious for four days. The damage was chiefly to the right side of the head, and there was a large scar over the right eyebrow. After the accident his R. vision failed and he found he could not shoot from the right shoulder. On this account he was transferred to the Ordnance Corps.

On examination the vision of the right eye was finger counting at 2 feet, no improvement with lenses. There was a punched out hole at the right macula, and the surrounding retina was slightly detached (Fig. 3).

CASE 4. J.S., schoolboy, aged 9 years.—Three years previously he was struck on the left upper lid by an air-gun pellet. After this he could not see well with this eye, and has never done so since.

Examination L. eye, less than 6/60, not improved. There was a circular punched out hole over the left macula, and a thin disc of tissue floating in the vitreous in front of it. The inferior half of the retina was detached. No other retinal hole found (Fig. 4).

My thanks are due to Mr. J. E. A. O'Connell for asking me to see cases 1 and 3 while they were under his care at Hill End Hospital.

A CASE OF FISTULA OF THE CORNEA. A METHOD OF TREATMENT

BY

J. B. McAREVEY

DUBLIN

DE SCHWEINITZ defines a fistula of the cornea as an orifice remaining after a wound, or more commonly because of the failure of an ulcer to heal. Fistula is an unusual sequel and when it does occur it is the central part of the cornea that is most often affected. De Schweinitz recommends to touch the mouth of the fistula with the point of the lunar caustic—and even to pare the edges and introduce a corneal suture. In Fuchs' Text-Book of Ophthalmology the line of treatment suggested is:—

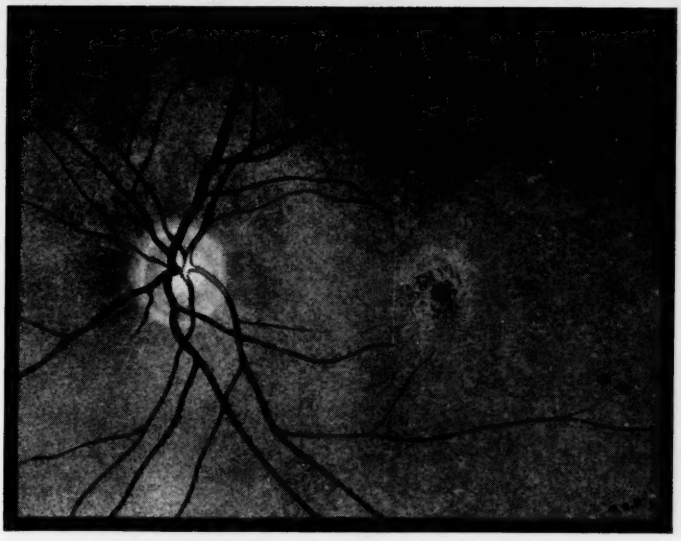

FIG. 1.

The left eye of E. L. after a wall had collapsed on him. Traumatic oedema of the macula.

FIG. 2.

Sergeant H. L. G. Pigmentary changes at the left macula probably following traumatic oedema.

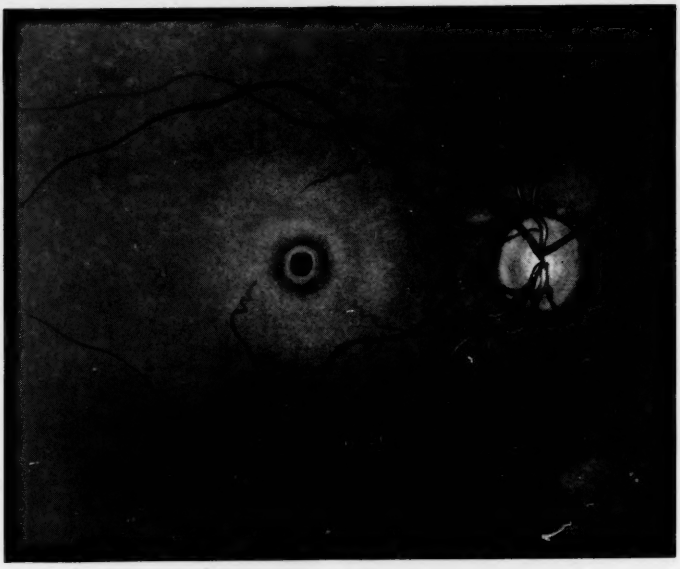

FIG. 3.

The right eye of J. S. four years after a severe head injury. Hole at the macula.

FIG. 4.

The left eye of J. W. three years after he had been struck in the left lid by an air-gun pellet. Macular hole with retinal detachment.

1. To avoid everything that might temporarily increase the ocular tension.

2. The use of miotics and a light pressure bandage.

3. If closure does not succeed a cauterization of the edges of the wound (this pre-supposes the presence of an anterior chamber on account of injury to the lens), again failing closure the use of a conjunctival flap having removed the epithelium, and as a last resort the removal of cicatricial tissue and implanting in the opening a piece of healthy cornea.

The presence of an anterior chamber has a bearing on the treatment; in the case I am reporting there was a definite hole through the cornea which appeared to have a uniform diameter of about a millimetre. To close the hole either a conjunctival flap or a graft would be necessary, the treatment with light pressure bandages for over a week having proved ineffective.

It occurred to me that as there was such a slight loss of tissue the swelling of the corneal stroma might close the hole and allow cicatrization to take place if the sides of the fistula were freshened up.

A man aged 27 years was working with metal and a piece flew off and hit him in the right eye. This patient was referred to me five days after the accident. Examination showed left eye normal. Right eye relatively quiet with slight watering, no anterior chamber present, contracted pupil, small central opacity of the cornea 2 mm. in size, mydricaine dilated the pupil well; X-ray negative, magnet negative, vision 3/60, fundus appeared normal, slit-lamp examination showed in centre of the opacity a hole in the cornea which appeared to have a uniform diameter of about 1mm., from the end of the fistula on posterior surface of the cornea Descemet's membrane radiated in folds. There was no anterior chamber and the lens was normal. On putting fluorescein into the conjunctival sac and washing away any excess, green circles started from the mouth of the fistula increasing in size as they passed towards the periphery of the cornea; fluorescein should be used as a diagnostic aid in doubtful leaking corneal wounds.

Under a local anaesthetic a series of perforations were made into the clear cornea just outside the opacity with a discission needle, these perforations were intended to enter the fistula at different levels, in addition three small incisions were made into the corneal stroma with a Ziegler knife—a fine probe was passed into these openings and pushed the corneal tissue towards the fistula. Both eyes were occluded with a light pressure bandage. In 48 hours the bandage was removed. The anterior chamber had reformed. The injured eye was kept bandaged for 5 days longer. After a week the patient was discharged from hospital.

Slit-lamp examination before leaving hospital showed what appeared to be a healed corneal wound with still some folding (greatly reduced) of Descemet's membrane, a normal anterior chamber, the eye free from inflammation, vision 6/18 and no apparent increase in the size of the original corneal opacity.

Summary.—A case of fistula or hole in the cornea is reported. Fluorescein is suggested as a confirmatory test in leaking corneal wounds and a method of treatment is described.

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AN OPERATION FOR ENTROPION OF TRACHOMA

BY

MAJOR C. COCKBURN, R.A.M.C.

IT is to be noted that the operation described below is essentially a slight modification of that described by Busacca. (*Arch. Ophthal.*, November 16, 1936).

As eye specialist to a Libyan prisoner-of-war camp during 1942, considerable experience in the treatment of trichiasis entropion has been gained, and it is considered justifiable to report on an operation which has been found to be most satisfactory.

About 60 cases, all Libyans, have been operated on. In all instances the disease had reached a chronic fibrotic stage with deformity of the upper tarsal plate and widespread trichiasis.

Anaesthesia.—Nerve block of supra-orbital and frontal nerves and local infiltration of eyelid with novotux and adrenalin.

A Desmarres' entropion forceps is carefully applied to the eyelid and the screw made firm to ensure adequate haemostasis. The skin is incised parallel to the lid margin and about 5 mm. above it. On either side the skin is well undermined to expose the fibres of the orbicularis muscle. The lower lip is freed right down to the cilia. The muscle fibres are then excised exposing the white tarsal plate. It is wise to remove all fibro-fatty tissue from the surface of the plate to facilitate further procedures. A linear incision is then made in the tarsus close to and parallel to its lower margin, down to but not incising the conjunctiva. A similar linear incision is made about 2 mm. above, and a strip of tarsal plate can then be readily excised. Even where the tarsal plate is thin, this procedure

FIG. 1.

Photograph taken immediately after completion of operation, showing arrangement of sutures.

FIG. 2.

Operation nine months ago for trichiasis entropion both upper eyelids.

involves very little difficulty. If the conjunctiva is inadvertently button-holed, it matters little. Special care must be taken that the strip of tarsus excised extends the whole width of the plate, and for this purpose it is often necessary to re-adjust the Desmarres' forceps. Four or five sutures are then inserted passing through the skin of the inferior lip of the wound and deep into upper lip of tarsus, and made taut. The ends of the sutures are left long and are fixed on the brow by sticking-plaster (Fig. 1). The upper lip of the skin flap is undisturbed. Depending on the degree of entropion, a wide strip may be excised or the sutures placed nearer to the cilia. It is not necessary to bandage both eyes. The sutures are removed in four days.

Recently, most of these cases have been re-examined, and I have good reason to be pleased with results. In no instance has there been a recurrence of the entropion and the cosmetic result has been excellent.

It would seem, therefore, that here is an operation which presents the following advantages:—

1. It is easy to perform.

2. It gives a complete and permanent cure of entropion.

3. It gives an excellent cosmetic result. In fact the slight ptosis present in all these cases is rectified, giving a rather pleasing, alert appearance (Fig 2).

For these reasons, it is considered that this operation might be more widely known and practised.

A MODIFICATION OF ARRUGA'S SPECULUM

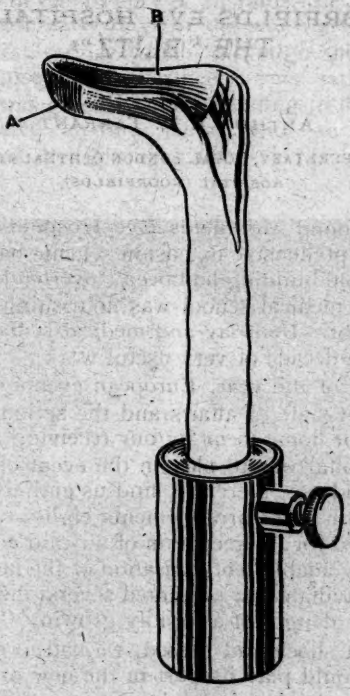
BY

HUMPHREY NEAME

LONDON

FOR operation on detachment of the retina, Arruga's speculum of plated metal was taken as a model and an instrument made of plastic material was designed 2 mm. wider than the original, so as to give a larger field within the hollow of the retractor for the application of diathermy in detachment of the retina.

The figure shows the general appearance of the speculum. The plastic material is roughened for a small area on its convex surface and for a larger area in the hollow of the concave side. Light enters from a small battery or through a transformer from the main



Neame's modification of Arruga's Speculum.

A. Outer surface } roughened.
B. Inner surface }

at the handle and is carried by internal reflection to the end of the instrument and is only dispersed when it meets the roughened surface. The retractor is, of course, quite free from heat, hence the proprietary name given to the instrument (Coldlite). The illumination of the area of the sclera requiring treatment is admirable and entirely obviates the presence of any shadows such as are inevitable to some extent in using a hand operating lamp. The greater width of the speculum is also an advantage and does not in the least make it difficult to use. The third and very definite advantage of this instrument is that it is a non-conductor of electricity.

This instrument is likely to supersede all other forms of retractor or speculum for this type of operation, for cases in which the area of the eye to be treated by diathermy is in the anterior part of the eye as well as in the posterior part.

MOORFIELDS EYE HOSPITAL IN THE "BLITZ"*

BY

ARTHUR J. M. TARRANT

SECRETARY, ROYAL LONDON OPHTHALMIC HOSPITAL (MOORFIELDS)

THE year 1938 found Moorfields Eye Hospital in a very happy position. A comprehensive extension scheme had been carried to completion; the old building had been "overhauled" at all points; the postgraduate medical school was flourishing and the hospital was free from debt. Both lay and medical staffs could justifiably look forward to a decade of very useful work.

In the autumn of the year, European events cast their shadow over this pleasant state of affairs and the seriousness of the situation was brought home to us by our receiving instructions as to the part the hospital was to play in the event of hostilities. The coming of war did not, therefore, find us entirely unprepared and in accordance with prior arrangements eighty-two beds were immediately reserved for the reception of air-raid and Service casualties. In the unavoidable reorganisation of the medical service, the problem of the civilian sick presented several difficulties, the chief of which was the danger of a rapidly growing " waiting list."

When the first shock had passed, we had to consider how best " Moorfields " could play its part in the new order of things. It is never easy to make decisions when facing a situation containing several unknown factors. As far as our own problem went, two facts cleared themselves from the background of " ifs and buts." The first was that we could not make much headway with our greatly reduced bed complement, and the second was that the hospital was in a vulnerable area. Both these facts pointed to the advisability of finding a temporary hospital outside London to supplement the work that could be done at " Moorfields." We were fortunate in being able to take over a small institution at Edgware and as early as October, 1939, we had converted it into a fifty-bed ophthalmic hospital, complete with an operating theatre. This country branch was able to deal adequately with cataracts, detached retinas, glaucomas and squints, leaving the eighty " eye " beds that we were able to keep at the main building to deal with accident and emergency cases. The out-patient attendances temporarily dropped during the uncertain days immediately following the out-

* Reprinted by permission of the Editor from *The Medical Press and Circular*, January 27, 1943. We are also much indebted to the Editor for the loan of the blocks for illustration.

break of war, but in the absence of air-raids, the numbers steadily increased. In those early days of the war we were thus able to carry on a hospital service which, although inevitably to some extent a compromise, was nevertheless very satisfactory. Some sense of security was given by the providing in the basement of a specially constructed air-raid shelter with an air-conditioning apparatus and auxiliary lighting plant.

FIG. 1.
. . . And so to Bed.

On a sunny Saturday afternoon in September, 1940, the Luftwaffe suceeded in firing the dockside district of London. In the evening the red fires of destruction could be seen from the roof of " Moorfields " as a terrifying spectacle. During the night the bombs came closer and nearby buildings were hit. Within three nights, two neighbouring hospitals received direct hits. It became necessary to reduce the number of " eye " beds at " Moorfields " for it was evident that only that number of in-patients could be

admitted, which, together with the required nursing staff, could be housed each night in the air-raid shelter, and so, in the winter of 1940-41, we settled down to a routine underground night life. Perhaps the best impression of that period can be got from the following account written at the time:—

"In this winter of 1940, the good people who work in the City of London go hurrying homewards before the twilight fades into night. For a while, a silence such as the city has not known for a century, settles on its ancient thoroughfares. It is a sinister silence into which at any moment may come the wailing of a siren, the droning of aeroplanes, the barking of guns and the destructive crump of bombs.

FIG. 2.

Good Night!

"At 'Moorfields,' where the work still goes on, we now have a definite 'night life.' Patients, house-surgeons, nursing staff, cooks, engineers, maids, porters, all sleep in shelters in the basement of the hospital and each evening, as a matter of routine, there is a steady, well-ordered trek to the 'dug-outs.' Come and watch

FIG. 3.

Matron has a last look round.

for a while! Nurses and porters are bringing patients from the wards. A little group has gathered, waiting for the lift, The woman in the wheeled chair is a case of acute glaucoma. Her eye condition has been precipitated by excitement due to air-raids. The man on the trolley was brought in as a casualty. He was operated upon this morning. A stream of 'walking cases' passes us, guided by a nurse. They are mostly the more serious type of treatment case. As you watch, one thing is certain. You cease to

be an onlooker: you simply must lend a hand. You find yourself taking the arm of an old fellow and saying: 'Come along, granddad.' You find yourself going back to help with a trolley or maybe to take the hand of a wee laddie who somehow manages to smile at you under his bandages. In the basement corridor, you catch up with the main body, in time to witness something in the nature

FIG. 4.
Treatment in the Air Raid Shelter.

of a minor miracle. For one moment you see a medley of trolleys, wheeled chairs, walking patients, nurses, porters, and maids carrying bundles of bedding. Then suddenly, as though someone had waved a magic wand, the corridor is clear, the patients are in their shelters, and the little table with its drugs and dressings is in its place. Over all is an air of calmness. We have gone to earth for the night! At six o'clock supper is served. In such unusual circumstances it could hardly fail to be a jolly meal, for it is typical

of these patients that they are ever ready to cover their personal anxieties with a veil of light-heartedness. When the meal has been cleared away, one or two dressings have to be done, and the patients are made comfortable for the night. Matron has a last look round and has a word for those who have not yet gone to sleep. In other 'dug-outs' those who are off duty amuse themselves in a variety of ways, but one by one go off to bed, and save for those whose duty it is to watch, this oddly-assorted little community settles down for the night."

So the work at "Moorfields" went on, the nightly air-raid warning sounding as a matter of course. From time to time we received air-raid casualties with eye injuries. It may be of passing interest to note how such injuries differed from those seen in ordinary civilian practice. The Senior Resident Officer of the hospital summarised the differences thus: "First, there was a large number of cases of gross traumatic conjunctivitis and keratitis that are unusual in civilian practice. Secondly, there were many instances of injuries due to glass splinters. It is interesting to note that a piece of glass in the eye could remain in it without causing any irritation and is better left alone if the eye is not inflamed. Thirdly, intra-ocular foreign bodies in these war-time casualties were very often non-magnetic. The patients were generally so extremely shocked that it was unwise to proceed to operation as quickly as one usually does in ordinary civilian work."

A very great demand was made on the hospital on the night of December 29, 1940, the night of the great fire of London. No less than 128 firemen were seen as casualties and all but four of them had corneal or conjunctival foreign bodies. One interesting thing about these cases was the intense degree of blepharospasm. This was due to a staining of the cornea following, no doubt, irritation by smoke. A temporary casualty department was set up in the comparative safety of the air-raid shelter, but as the number of injured firemen increased, it became impossible to cope with them under temporary conditions. The work, therefore, had to be continued in the hospital's usual casualty department, a ground floor room surrounded by glass. Although following the fire raid high explosives were being dropped, surgeons and nurses worked continuously through the night. All these firemen were cured within a few hours, none of them had to be admitted as an in-patient, and only one required attention for more than four days.

On January 11, 1941, "Moorfields" was badly damaged by blast. It is an astonishing sight to see blast damage on a large scale. That which has stood in its customary place for years is shattered in the twinkling of an eye. Rooms and corridors throughout the hospital were strewn with splintered wood and glass. The patients were below in the air-raid shelter. All of them

were serious cases. In such circumstances it need hardly be stressed that the demeanour of the nurse reacts immediately upon her patients. Whilst the raid was in progress, we saw a nurse,

FIG. 5.

The morning after a raid.

half-sitting on the mattress of an elderly woman, with her arm reassuringly round the patient's shoulder. (If this were "unprofessional" we are sure that Matron forgave her). Together

they were looking at a book. If we remember rightly, it was called "The Year's Photography" and was a collection of pictures. Each one was being quite seriously discussed. With such an example, it is needless to add that everybody seemed perfectly calm in that section of the shelter. When the day broke, the staff of the operating theatre made their way over the debris to the theatre and found it apparently in a hopeless state. They worked all through the day until a minor miracle had been achieved and order restored out of chaos. Finally, men were found to put temporary coverings over the window spaces and by the evening the theatre was ready for use again and an operation was actually done. "First-aid" repairs were started immediately. After ensuring that the work of "Moorfields" could still go on, we put back the front door and drew up a rough priority list for temporary repairs which were all eventually carried out. The night of April 15, 1941, was a night of fires in the neighbourhood of the hospital. Several adjacent buildings were burnt out and at one time it seemed impossible for "Moorfields" to escape the tongues of fire. The incendiary bombs which fell on the building were extinguished by the engineering staff and by the fire watchers on duty. On May 10, the hospital was again damaged by blast and repair work started all over again!

In any account of these days at "Moorfields" a word of praise must be given to the kitchen and domestic staffs. They are so easily forgotten and yet the part they play is no small one. On a number of occasions there was neither electricity nor gas, yet these unsung heroines never failed to produce a meal of some sort at the right time. For one spell it meant the improvisation of a coke fire in the courtyard of the hospital.

Perhaps the writer, who is a layman, may also be allowed to pay tribute to the resident medical staff and to the nursing staff of the hospital for their conduct during this trying period. It is readily admitted that the stories told here lack the lustre of the exceptional heroic deeds of which we hear from time to time. They do, however, emphasise the sense of service which animates the nursing profession. This high conception of their calling which in peacetime finds expression in a nurse's cheerful devotion to duty, explains why in war-time she will unhesitatingly shield her patients, if need be with her own body.

ANNOTATION

Eye Conditions among Pupils in Schools for the Blind in U.S.A., 1940-41

A report on eye conditions among pupils in schools for the blind in the United States, 1940-41, was supplied by Miss C. Edith Kerby for the committee in statistics of the blind, and is published in "Outlook for the Blind, and The Teacher's Forum," Vol. XXXVI, December, 1942. This report forms the eighth of a series of annual analyses of data on eye conditions among blind children of school age in the States. It covers 75 per cent. of the present enrollment in the elementary and secondary grades of the residential schools for the blind and day classes in the public schools serving 39 states and the district of Columbia. Slight changes in distribution have occurred, but not of sufficient significance to warrant the conclusion that there have been any marked changes in the characteristics of the groups studied during the year in question. There has been a notable improvement in the completeness of the records in some schools, those which have been included in the study for some years, while in new groups there is more incompleteness. Whilst "not reported" cases now constitute a very small proportion (less than 3 per cent.) of the total for items such as present age, age at onset of blindness, amount of vision remaining and nature of eye affection, the aetiological factor in blindness remains totally unknown in 10 per cent. of cases and is identified only as "pre-natal origin, cause not specified" in an additional 38 per cent. of the total cases up to the present time.

Miss Kerby makes out a good case for much more intensive study of the history of each case, and for this service the schools for the blind should accept responsibility.

"It is discouraging to note the number of eye examination records on which recommendations for treatment or operation have been noted, and yet these pupils have remained in the schools for the blind year after year, with no indication that the recommendations have been carried out." And there is "the problem of the continued presence in the schools of children (amounting to nearly 12 per cent. of the pupils) who apparently have too much vision for education by tactual methods." Infectious diseases are responsible in this report for 23·9 per cent. of the blindness among children. The chief are ophthalmia neonatorum 10·9 per cent., syphilis 5·0 per cent., and meningitis 1·8 per cent. The true figure for syphilis is probably much higher. Trauma accounted for 7·9 per cent., and in 4 out of 10 of the injury cases blindness was due to sympathetic

ophthalmitis. At least 13·5 per cent. of the blindness, in children of school age is due to heredity.

"The public should be educated regarding the potential danger to the eyes in cases of systemic disease involving either degenerative changes or nutrititional deficiencies, as well as in infectious diseases." Miss Kerby notes that 521 children in the schools for the blind during 1940-41 are there because of ophthalmia neonatorum, and 230 children are there because of syphilis pre-natally acquired. There is a folding table of the causes of blindness among pupils in the schools and day classes for the year in question, and other tables show the names of the schools with totals of pupils in each; distribution by amount of vision remaining; age distribution; and age at onset of blindness, all of which merit careful perusal.

Miss Kerby is to be congratulated on this excellent report.

LIGHT AND VISION

[*Abstract of an Address by* SIR JOHN PARSONS *to the Illuminating Engineering Society on May 11, 1943.*

The Illuminating Engineering Society was founded by Mr. Leon Gaster in 1909 for the purpose of promoting "good lighting," i.e. lighting "adequate and suitable" for the given performance. It was designed to represent all aspects of the subject, and its members include gas and electric light experts, physiologists, ophthalmologists, architects, and others. The writer is an original member, past president, and fellow of the Society.]

AT the time of its inception there was little guidance in lighting matters from ophthalmologists and physiologists, but it must be remembered that information on the physical side was also vague. Although engineers were becoming accustomed to foot-candles the few instruments for measuring illumination were primitive and cumbersome. There were as yet few records of the illumination actually provided in different classes of buildings. At that time, too, the conception of brightness in terms of candles per square inch was little understood, and there were no quick and convenient means of measuring the brightness of sources.

In those days, as Sir Francis Goodenough has said ("Illuminating Engineer," Vol. xxv, p. 276, 1922), "Technicians were somewhat apt to devote themselves to the perfecting of their tools

rather than the objects they served. For many years the chief task had been that of producing light economically and abundantly." One may compare this with the printers, whose object seemed to be to produce a beautiful page, with no regard for legibility. Since then there has been a marvellous advance in the efficiency of illuminants, so that undue stress has been laid upon physical intensity. None the less in the first paper contributed to the Society on Glare (" Illuminating Engineer," Vol. iii, p. 99, 1910) I drew attention to the dangers of excessive light and the importance of the distribution of light in the field of vision.

Further, as Dr. Wright has reminded me, the average illuminating engineer is scared stiff of anything at all academic about the eye or the visual response; just as, in my own experience, the average medical student is scared stiff of mathematics and analytical optics. Being confronted with the chaos of metre-candles, foot-candles, equivalent foot-candles, lamberts, lumens, and photons, not to mention daylight factors and brills, may account for the apathy of ophthalmologists.

These opposed limitations in mental outlook are responsible for many of the errors and shortcomings of both physicists and physiologists, and for the lack of liaison between them. As I have often pointed out physicists tacitly ignore the physiological basis of all their measurements, hiding their heads under the convenient term " errors of experiment," and resorting to statistical methods to condone their ignorance. On the other hand there are many reasons into which I cannot enter now which militate against physiologists devoting themselves to research on the special senses; and psychologists who ought to make these subjects their own prerogative, are nearly without exception ill equipped for the purpose. Nearly all the experiments which have been made by psychologists and many by physiologists are almost useless owing to the absence of accurate quantitative data. The experiments of von Kries and his pupils on colour vision and those by Lythgoe on peripheral vision, etc., are noteworthy exceptions.

The problems with which this Society is concerned are preeminently such as cannot be segregated into purely physical and physiological compartments. The physicist has been forced to open his eyes to physiological facts. An early example is the " deviations from Newton's law of colour mixtures," as König called them, which are due to the difference of scotopic from photopic vision. On the other hand, one has only to recall the names of Newton, Thomas Young, Clerk Maxwell, Helmholtz, König, Abney, and many others down to the present day to realise that we owe most of our knowledge to physicists.

The Illuminating Engineering Society was founded for a definitely utilitarian purpose—that of promoting " good lighting."

Good lighting is sufficiently well defined in the Factory Lighting Committee's report as " adequate and suitable " for the given performance. One is almost startled to find that the problem is primarily and fundamentally physiological, for adequacy and suitability depend upon the performer's capacity. Ophthalmologists knew that prolonged near work under low illumination and unsuitable conditions led to fatigue, " eyestrain," headaches, and bad health. Industrial fatigue is difficult to measure, though much valuable work was done by the Industrial Fatigue Board of the Medical Research Council, using output as the criterion. The retina appears to be insusceptible to fatigue in ordinary conditions, and eyestrain is chiefly due to fatigue of the extra-ocular muscles; hence the importance of measuring convergence capacity and latent squints. Even more potent in causing eyestrain are uncorrected errors of refraction. It is only comparatively lately that examination of the eyesight of school children and the correction of their refractive errors have become compulsory, largely due to the pioneer work of Dr. Kerr and Mr. Bishop Harman for the London County Council. May we not hope that the sight of factory workers will be similarly catered for? A striking example of the value of the application of knowledge of this kind is the success of Mr. Weston's efforts to ameliorate the strain of very fine work by the use of suitable convex glasses combined with prisms.

Since good visual acuity is the *sine quâ non* of clear vision the effects of the quantity and quality of the illumination early attracted attention. Visual acuity is measured scientifically by the minimum visual angle subtended at the nodal point of the eye by two point sources necessary for resolution. So far as intensity is concerned we know now that the acuteness of vision becomes progressively better up to, and even beyond, 1,200 ft.c. Unfortunately, as Lythgoe has pointed out (Trans. I.E.S., Vol. i, p. 4, 1936), it was not sufficiently recognised in the early days that visual acuity is proportional to the logarithm of the illumination; so that on the linear scales then prevalent it appeared that there was little improvement above 3 or 4 ft.c. It is noteworthy that until the Council of British Ophthalmologists took the matter up in 1919 there was no standard fixed for the illumination of Test Types, and even so it was fixed at too low a level, which had to be increased in a subsequent report (1935).

Even scientifically disposed ophthalmologists are liable, like other scientists, to simplify their problems unduly, and consequently to attach undue importance to isolated details. Thus, little attention was paid in the above determinations to the surrounding illumination. It has since been found that this has a very marked effect. To get the best result the surrounding illumination should be only slightly less than that of the test object, book, or work. If

it is much less visual acuity falls off after a maximum at about 40 e.f.c. illumination of the test object, and if it is quite dark the maximum is reached at less than 20 e.f.c.

The Society deserves to be congratulated upon the issue of the I.E.S. Code of Illuminations adequate and desirable for different classes of work. It is largely founded upon empirical data, and much research yet remains to be done; but that it filled a long felt need is shown by the hearty reception it has had.

Taking together the now justified predilection for higher illumination for reading and work, and the economic availability of greatly increased artificial illumination it is not surprising that people demand these higher illuminations, especially when we find that if the general illumination exceeds that of the test object visual acuity will be seriously diminished. Whether the satisfaction of this desire is being carried to extremes with deleterious results is a matter for future investigation; but the suggestion that the demand for high illumination is due to general deterioration of sight in the population is entirely unsupported by any valid evidence.

The effect of the surrounding illumination on visual acuity is one of the many characteristics of the eye as an optical instrument of precision which are so exasperating to the physicist. The retina is a part of the brain, and it is no surprise to the physiologist that there is interaction between the different parts. Much light has been thrown in recent years on this subject by the investigation of the electrical responses to stimuli by Adrian and Matthews, Hartline, Granit and others, and by the researches of Lythgoe and Tansley. It has long been known in the manifestations of simultaneous contrast. In its most blatant form excessive contrast plays a preponderating part in glare, as was indeed fully recognised in the earliest days of the Society. It was to the discussion on Glare, initiated by my paper at the first meeting that Prof. L. Weber of Kiel contributed recommendations which were substantially adopted by the Factory Lighting Committee and have even now been little modified. He advocated (1) a limit to brightness (about that of a candle flame) and (2) the location of sources outside the 30 deg. angle at the eye. But he also approached near to the root of the matter in suggesting that (3) the ratio of the brightness of the source to that of the surroundings should not exceed 100 to 1 —a value deduced from daylight conditions.

The earlier researches aimed at a satisfactory explanation of glare by Cobb and others were disappointing, and we owe it to Dr. Stiles that it has been put upon a more satisfactory basis, though I am sure he would be the first to admit that we have only touched the fringe of the subject. Recognising that glare is largely due to scattered light, *i.e.*, light in the wrong place, he conceived the

brilliant idea of translating its effects into those produced by flooding the whole area of the retina with uniformly distributed light; thereby enabling the results to be expressed in comparable quantitative terms.

We have at present no means of detecting glare directly in the physiological sense. We have therefore to fall back on indirect and empirical methods of controlling the circumstances which, it is conjectured, will give rise to glare; for example, by limiting the brightness of sources, or specifying for them positions outside the immediate range of vision. Possibly some simplification might be effected by some general rule specifying the desirable contrast or brightness ratio between object and surroundings, which would not only act as a safeguard against glare, but also ensure conditions favourable to the powers of perception of the eye. In this connection one must not forget glare due to specular reflection from polished material; nor the inconvenience and occasional danger of harsh shadows. The precise definition of shadow offers as many difficulties as the assessment of glare.

With regard to the quality of light our eyes are not only capable of adaptation to the enormous range from 0·00001 to 10,000 ft.c., but also to considerable variations of spectral constitution. Phylogenetically eyes have obviously been adapted—in the evolutionary sense of the word—to sunlight, but there is no evidence that artificial sources of so-called " white light," composed of practically continuous spectra, *per se* cause any harmful effects. These are generally due much more to the distribution of the light than to either quantity or quality. It is true that they nearly all, including the sun, emit ultra-violet radiation, but the atmosphere usually acts as an efficient filter in the case of the sun, and artificial sources are enclosed in glass which is equally efficient. The deleterious effects of ultra-violet light in everyday life have been grossly exaggerated. Similarly, the craze for " daylight " lamps, apart from special colour-matching processes, etc., is quite unwarranted.

The effects of monochromatic light and of sources like mercury vapour lamps which emit discontinuous spectra, and of those with intermittent emission, especially when the cycles per second are few, are more debatable.

The phenomenal advance in the development of discharge lamps using fluorescent substances—an advance due largely to the G.E.C. Research Laboratory under the aegis of Mr. C. C. Paterson, F.R.S.—emphasises the importance of colour vision and its defects. The work of Guild and others on Colorimetry resulted in 1931 in the standardisation of colour mixture curves by the International Commission of Illumination—a fitting sequel to the standardisation of the equal energy luminosity curve in 1924 by the

same body. Together they form perhaps the most noteworthy advance in photometry of recent date. Neither has the theoretical side been neglected. Dr. W. D. Wright's reinvestigation of the so-called sensation curves by more refined methods has corrected some discrepancies in the work of König and Abney; and by using the method of binocular matching, in which adaptation effects induced in one eye are measured by comparing vision in that eye with vision in the constantly dark-adapted other eye, he and others, *e.g.*, Schouten and Ornstein, Pitt and Craik, have elicited new facts of theoretical and practical value.

Considering the great prevalence of colour blindness in different degrees—at least 5 per cent. of the male population are badly colour-blind—it is remarkable how little this disability seems to affect every-day life. Yet many of these are constantly engaged in delicate photometric observations. In most such tests difference in hue of the test objects is reduced to a minimum, so that the estimate is one of equality of brightness in two white lights. There is some evidence that pure brightness discrimination is actually better in the colour-blind than in the normal—a fact which has been adduced as a factor in the excellence of the etchings of Méryon. I suppose that in the grosser forms of heterochromatic photometry the results are always controlled by the flicker method. We may hope that Dr. Wright's researches will throw further light on the luminosity curves of the colour-blind. Even in colour matching, however, such colour-blinds as conform with the Trichromatic Theory would be expected to accept the normal matches. In fact, they do so, but, unfortunately, as Prof. William Watson pointed out, they will also accept considerable deviations from the normal match.

Until the outbreak of the war and the initiation of the " black-out " illuminating engineers paid little attention to the peculiarities of vision under very low illuminations. Probably their first experience of these was due to anomalies in photometric observations caused by the Purkinje phenomenon. Previous to the " black-out " town dwellers had little experience of complete dark adaptation, and it is therefore not surprising that they should be found to be psychologically relatively night-blind compared with country dwellers. Attention was first seriously devoted to the subject when exhaustive researches were made to improve street lighting and put it on a satisfactory scientific basis. The Factory Lighting Committee were faced with the problem of setting up standards for the illumination of passages in factories, non-working areas, dock-yards, and so on. These were fixed as follows: (*a*) over interior parts of a factory other than working areas, 0·1 ft.c. (1915), subsequently increased to 0·1 ft.c. to 0·5 ft.c. (1938); (*b*) over open yards, etc., not less than 0·05 ft.c., subsequently increased to form 0·05 ft.c. to 0·1 ft.c. In the present circumstances the conditions

of vision under moonlight and starlight illumination are literally of vital importance to airmen and other combatants.

None of the standards hitherto set is based on really satisfactory scientific evidence, partly on account of the ignorance of physicists and illuminating engineers of such facts of the physiology of scotopic vision as are known, but much more because of the ignorance of the physiologists themselves. A vast amount of very valuable work has been done on night vision for the Services, especially the R.A.F., and, while it has been of immense practical utility, it has but served to reveal the extreme complexity of the subject.

Dark adaptation is characterised by a phenomenal increase (from about 0·00001 ft.c. to 10,000 ft.c.) in the sensitivity of the retina, which rises rapidly during the first ten or fifteen minutes and reaches a maximum in about three quarters of an hour. This rise in sensitivity shows some variation in the normal population, but there are pathological cases in which it is delayed and defective and may be very bad.

When a spectrum of low intensity is viewed by the dark adapted eye it appears as a colourless band, having a luminosity curve which is of the same general type as the photopic luminosity curve, but having its maximum at about 500 $\mu\mu$ instead of 550 $\mu\mu$. This shift of the luminosity curve accounts for the Purkinje phenomenon, in which the apparent brightness of the short wave-lengths is much enhanced compared with the longer wave-lengths.

Visual acuity, even under photopic conditions, falls off very rapidly as the fixation point is shifted from the centre, *i.e.*, direct vision to the periphery.

The fovea which is the site of highest discrimination or visual acuity, subtends a visual angle at the nodal point of the eye of 60′ to 70′; the macula is oval, with the long axis horizontal, subtending a visual angle of 4° from above down and 12° across. The deeper layers of the retina not only conduct impulses directly to the brain, but have intercommunications which permit of modifications in the responses of adjacent areas.

It is certain that the rods contain a photochemical substance which subserves the transformation of the physical stimulus into a nervous impulse. This substance, rhodopsin or visual purple, appears to be absent from the cones, where it is almost certainly replaced by a similar photochemical substance or substances. Visual purple is bleached by light, and its absorption curve is identical with the scotopic luminosity curve, so that it is practically certain that it is the physiological basis of vision under very low illuminations. Dark adaptation is due to the accumulation of visual purple, and since rods are absent in the greater part of the macula, it might be expected that this area would be night-blind.

That this is indeed the case was discovered long ago by astronomers, who found that a star of very low magnitude could be seen only by looking at it eccentrically, so that the image fell upon a parafoveal spot. The macula is not, however, wholly incapable of dark adaptation: such as it possesses is indeed slight, but rapid in development.

The researches of Lythgoe and Goodeve, and of Wald have thrown much light on the constitution and properties of visual purple. It has long been known that in certain cases of malnutrition, *e.g.*, xerophthalmia, night blindness is a cardinal symptom. It is now known that the presence of vitamin A is essential to the regeneration of rhodopsin. As a result a vast amount of work has been done in the hope of improving night vision by administering large doses of vitamin A. The evidence seems to prove that this has little or no effect on normal subjects, but that if the defect is due to malnutrition the effects are striking.

König showed many years ago that if the effects of illumination on brightness discrimination are plotted on a logarithmic scale two straight lines of different slope result, the kink occurring at about 0·25 metre-candles (0·023 ft.c.). This has been confirmed by flicker photometry: and the same curves are obtained with the effects of illumination on visual acuity. There can be little doubt that, in accordance with the Duplicity Theory, the scotopic part of the curve is due to rod vision and the photopic to cone (± rod) vision.

The transition from day vision to night vision therefore occurs when the illumination decreases to less than that provided by the full moon, about 0·023 ft.c., night vision being fully established at illuminations of less than 0·01 ft.c. Above the upper limit photopic, foveal, cone vision will be in the ascendant; fixation will be good, and visual acuity good. Below the lower limit scotopic, rod vision will be used; fixation will be bad, parafoveal, and fluctuating owing to rapid fatigue of the rods, and visual acuity will be bad. At the intermediate levels fixation will be fluctuating, and it is likely that vision will be worse than might be expected owing to rivalry between the two systems.

But there are other factors which make the complexity of scotopic vision much greater. The chief are the size of the area stimulated and the contrast or brightness discrimination at these low levels of illumination. For visual angles up to about 40′ to 50′ Riccò's law holds good for central vision; *i.e.*, at the threshold *the product of the area into the light intensity is constant.* The threshold luminous flux is about $0{\cdot}2 \times 10^{-12}$ lumen for extra-foveal, and 8×10^{-12} lumen for foveal vision. For larger areas the total luminous flux is no longer alone responsible for the threshold. According to Piper the product of the threshold intensity into the square root of the area is constant, but this is only valid for

white light and objects subtending a visual angle up to 10° (Henius, Fujita). Similar laws hold good for the detection of the orientation of Landoldt's broken ring (C), or an E; but this only shows that these are really tests of brightness discrimination, and are quite unsatisfactory tests of true visual acuity or form vision—at any rate at low intensities of illumination.

Brightness discrimination depends upon the area even at normal levels of illumination. With a photometric field of 5° the eye has to work with regions of the retina which differ in sensitivity, and more consistent results are obtained when the visual angle is reduced to 2½°. At 1° vision is foveal and purely photopic, and there is no kink in the logarithmic curve. The transition occurs at about 0·03 e.f.c., the order of brightness provided by the full moon, and is responsible for the low values of $^{\Delta}B/B$ which are obtained with large fields at intensities far below those at which discriminating capacity vanishes with smaller areas. It is important to remember that $^{\Delta}B/B$ expresses only relative brightness difference in terms of stimulus values, and is only valid as an expression of physiological apparent brightness difference or true contrast over the limited range in which the Weber-Fechner law holds good. The difficult problem of brightness and contrast is discussed in an excellent paper by Hopkinson, Stevens, and Waldram (Trans. I.E.S., Vol. vi, p. 37, 1941). The discordant results obtained by different observers are largely due not only to the differences of size and shape of the test areas, but also to the size of the illuminated surrounds. When the test field is exposed for only a short period of time a higher percentage of contrast is needed to produce a just noticeable difference.

Hopkinson, Stevens, and Waldram point out that when the adaptation level is reduced the instantaneous visual range of appreciable brightness is not symmetrically disposed about the adaptation level, as is approximately the case at the normal levels. "The eye can then distinguish brightnesses greater than that to which it is adapted, but not much to lower brightnesses"—a fact of great significance in street lighting and aeroplane spotting. It is also of considerable importance in the measurement of low values of illumination and brightness, a subject dealt with by Dr. Walsh (Trans. I.E.S., Vol. vi, p. 117, 1941). There is little reference to physiological conditions in this paper, except that in speaking of the Purkinje phenomenon the author says that "one can easily live in a fool's paradise,". It seems to me that, in the present state of knowledge, the same remark applies to making measurements in which "the range introduced is 0·0005 to 0·3 ft.c., and the corresponding limit of error laid down is 20 per cent. + 0·0001 ft.c." (Specification for Portable Photometers (Visual Type), 230. 1935). Fortunately, Dr. Walsh realises that "the necessity

for measuring apparent brightness, rather than brightness based on a physical scale, is perhaps the most striking of these new developments."

The lowest level at which consistent data as to the influence of contrast on visual acuity have been obtained is 1 ft.c., which is much greater than that prevailing at night; even for this illumination for less than 10 per cent. contrast large increases of size have to be made. Riccò's and Piper's results indicate the decreased importance of contrast at low intensities. Their results also show that in using field-glasses at night, with visual angles between 10′ and 2°, the size of the exit pupil, and therefore the amount of light gathered, is more important than the coefficient of magnification.

[The speaker then reviewed some of the activities of the Society in the past: *e.g.*, lighting of schools and libraries (1913), the lighting of factories and workshops, eyestrain in cinemas (1920), the I.E.S. Code of Illuminations).

The main functions of the Society have been well catered for in the past, and the formation of provincial Centres and Groups has thoroughly justified itself by arousing local interests and attracting attention to the needs of local industries. But "these things ought ye to have done, and not to leave the other undone," and the amenities of everyday life might be considerably improved if more detailed attention were paid to such problems as the distribution, quality, and aesthetic effects of domestic lighting. The experience of the Factory Lighting Committee has shown that problems of this kind are not so simple as might be thought. Many of the inconveniences which we grumble at but put up with —rooms in which it is almost impossible to read or sew with comfort, bedrooms with half a dozen mirrors in none of which it is possible to see to part one's hair or shave—might be eliminated or ameliorated if architects would take the trouble to learn from illuminating engineers and insist upon builders carrying out their instructions, such as putting in the lighting points where they ought to go, and not where the builder finds it most convenient for himself. The definition of the edges of steps and stairs and their lighting still seems to me to be unsatisfactory. Perhaps these and other similar problems might be referred to an Architectural Committee: but what I think is much more necessary is the establishment of a standing Physiological Committee.

ABSTRACTS

MISCELLANEOUS

(1) **Julianelle, L. A. and Smith, J. E. (Missouri).—A statistical analysis of clinical trachoma.** *Amer. Jl. of Ophthal.*, p. 158, February, 1943.

(1) **Julianelle** and **Smith** present a statistical analysis of 1,000 patients with trachoma graded according to MacCallan's classification into types I, II, III and IV.

About 15 per cent. belonged to type I or early trachoma, the majority of these complained of no symptoms and were detected fortuitously during routine inspection for prevention and control of trachoma in different communities.

In all types heavy secondary infections were uncommon, however in type II group there were three patients in whom gonococcal infection was superimposed on the trachoma, while in three other patients spring catarrh occurred as a complication.

A relatively large number of patients required repeated courses of treatment following recrudescence of the disease, that is to say that treatment had arrested but not cured the disease.

The source of infection was carefully studied in the 1,000 patients; in 387 it was undetermined, in 368 it was familial, and in 245 it was non-familial.

A cardinal criterion in the clinical diagnosis of trachoma is the eventual neovascularization of the upper fifth of the clear cornea or pannus, however, in 37 per cent. of the type I patients this was absent. In these cases the diagnosis was made by the future progress of the condition, presence of trachoma in other members of the family, occurrence of inclusion bodies in stained preparations of conjunctival epithelial cells, and finally, transmission of experimental trachoma to monkeys following inoculation of conjunctival scrapings. The authors estimate that within a month following infection up to 95 per cent. of patients already exhibit obvious pannus.

Among the complications described by the authors entropion was noted in 12 per cent. of all cases, by far the larger number occurring in the type IV group.

A. F. MACCALLAN.

(2) **Martin, J. D. and Mabon, R. F. (Atlanta).—Pulsating exophthalmos.** *Jl. Amer. Med. Assoc.*, January 30, 1943.

(2) The majority of the cases of pulsating exophthalmos are unilateral, usually follow trauma and are the result of communication between the cavernous sinus and the internal carotid artery. **Martin**

and **Mabon** describe five cases and review all reported cases. A reminder is given that patients with this pathological condition do not always exhibit pulsation or thrill; a bruit, however, is always present.

A. F. MACCALLAN.

(3) **Dubois, H. F. W. and Fischer, F. P. (Utrecht).—The effect of low pressure on the eye. (Ueber die Wirkung von Unterdruck auf das Auge).** *Ophthalmologica*, Vol. CII, p. 164, September, 1941.

(3) **Dubois** and **Fischer** followed up the work of Kukan in investigating experimentally the effect of low pressure on the eye. They show that the intra-ocular pressure is not increased by Kukan's method, but the extra- and intra-ocular venous pressure is reduced. Furthermore, the low pressure causes an increase in the volume of the globe and a change in the quality of its wall, thus increasing rigidity. Kukan's apparatus is shown to be useful not only for clinical dynamic rigidity determination but also for ophthalmoscopic measurement of the fluid pressure. This also applies to the Lindberg ophthalmo-dynamometer. Only Wessely's method renders it possible to increase the intra-ocular pressure by low pressure.

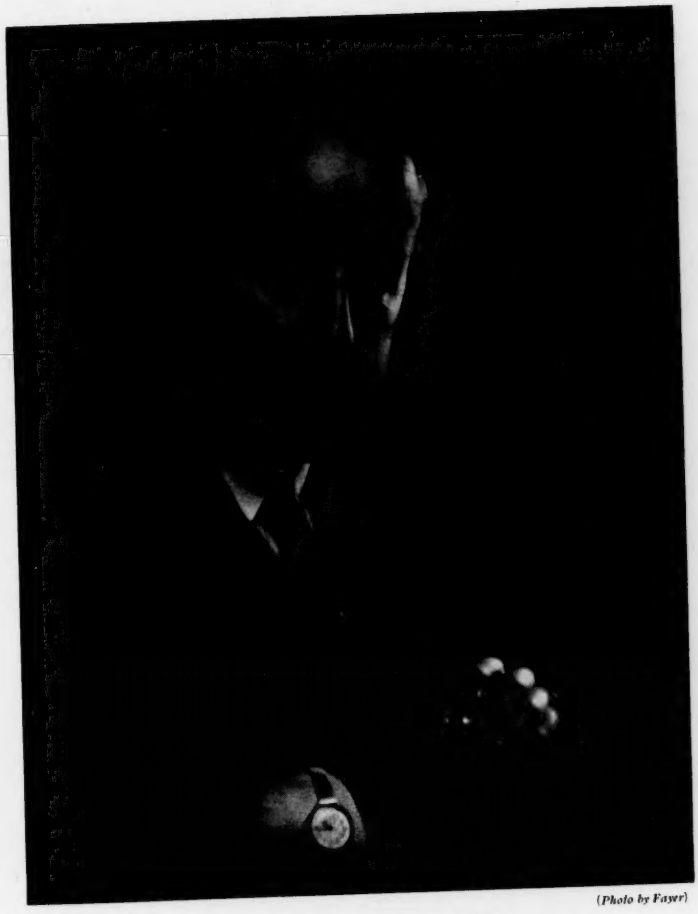
ARNOLD SORSBY.

OBITUARY

LESLIE JOHNSTON PATON

THE death of Leslie Paton on May 15, 1943, after a long and trying illness is a heavy blow to British, and indeed, to international Ophthalmology. To us of the British Journal of Ophthalmology it comes as a very sad wrench. Our older subscribers will recall the fact that we were barely three months old in the spring of 1917 when Mr. Jessop, who had been the mainspring of our foundation, died. It was a critical time but Lawford and Paton took on the management of our affairs and steered the ship to safety. The fact that we have never looked back since is a tribute to the unselfish work which Paton undertook. He was mainly concerned with our financial affairs. He once disclaimed to the present writers the possession on his part of great financial ability, but there is no doubt that his work put our financial position on a sound basis.

Paton was not one to rush into print on the slightest provocation, and when he produced a paper it was the result of long and careful cogitation. In a sense, this was to be regretted, because at least



(Photo by Fayer)

LESLIE JOHNSTON PATON

1872—1943

two discoveries might have been placed to his credit had he written about them earlier. The first was the Foster-Kennedy syndrome of optic atrophy in one eye combined with papilloedema in the other, which he mentioned to Sir William Gowers, who described it in a lecture. Paton's priority in this matter has been generously acknowledged in America, but is not so well-known here. The second was the organism responsible for angular conjunctivitis, which was isolated by him before the publication of the results of Morax and Axenfeld. But his literary output, if restricted, was of a very high standard and three papers of his are of outstanding importance. His work on the pathology of papilloedema in association with Gordon Holmes, has been accepted as final everywhere. Some years ago he published in our columns a masterly review of optic atrophy in tabes, and his paper on the demyelinating diseases accompanied by optic neuritis, which he delivered at Glasgow on the award of the William Mackenzie medal, was another noteworthy performance. It was peculiarly appropriate that he should have received the medal at Glasgow, for from thence he had his early schooling, and it was from Glasgow that his wife came. She was indeed his loyal helper, who in his early days supplied the encouragement and optimism without which no man, however great, can succeed, and in his later days saw to it that he was not overworked. It was also her hand which was responsible for drawing many lucid diagrams which illustrated his lectures and papers.

Leslie Johnston Paton was born in Edinburgh in 1872, the second son of the late James Paton. After leaving Glasgow he went to Cambridge as a scholar of Caius College and he had a distinguished academic career. He then returned to Glasgow, started training in Medicine there and completed his medical education at St. Mary's Hospital, where he served as house surgeon and Demonstrator of Anatomy before being chosen assistant ophthalmic Surgeon to the Hospital in 1902. His senior colleague was the late Henry Juler. In due course he succeeded to the senior post and on retirement became consulting Ophthalmic Surgeon and later Vice-president of the Hospital, the last being an office of which he was especially proud. Paton worked with Marcus Gunn for many years, both at Moorfields and at the National Hospital, Queen Square. In 1907 he became Assistant Ophthalmic Surgeon at Queen Square, and it was here that he laid the foundation of his international reputation in the field of ophthalmic neurology. He became president of the Ophthalmological Society of the United Kingdom in 1929, and president of the Section of Neurology of the Royal Society of Medicine in 1930. He also served as Treasurer of the English Speaking Ophthalmological Congress held in London after the last War, and had been Chairman of the Council of British Ophthalmologists and Treasurer of the International Ophthalmological

Council. Paton was an honorary member of many foreign Ophthalmological Societies, and an honorary fellow of the American Medical Association.

Tall and imposing of aspect Paton possessed a fund of humour and a very kind heart. He had an enormous private practice and was beloved by his patients, but in spite of the keen interest he took in his work, he did not allow this to monopolise his life. Although he was happy in the consulting room, hospital or operating theatre, he could take six weeks away from it all every summer and enjoy every minute of his holiday. There were few subjects on which he was not an authority and his wide reading and tenacious memory made him a formidable opponent in any general knowledge contest. He was also a phenominally quick reader and could get through four novels a day if laid up in bed with nothing else to do. Scottish to the core, he was a good golfer and fisherman. He kept his kindly Scots accent throughout life and many will recall his humorous statement in his presidential address at the Ophthalmological Congress, that, though born and bred in Scotland, he had lived long enough in London to make himself understood by southerners. He was in the true sense of the word a whole man, his entire character lit by the lamp of human kindliness, and a wish to help his fellow men.

To his widow and daughters we would offer our heartfelt sympathy in their great loss.

NOTES

Honour IN the birthday honours' list we are pleased to see the name of Mr. H. L. Eason, who becomes a Knight Bachelor.

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Appointment MR. ARNOLD SORSBY has been appointed research professor in Ophthalmology at the Royal College of Surgeons and at the Royal Eye Hospital for a period of five years.

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Special Notice WE are asked by the Ministry of Information to state that the fact that goods made of raw materials in short supply owing to war conditions are advertised in this journal should not be taken as an indication that they are necessarily available for export.